right jugular vein 5 grains were injected, respiration suddenly became difficult, accelerated, and spasmodic, with convulsions and tetanic extension and drawing back of the head, and death occurred in seven minutes.

Two grains of solanin were injected into the right jugular vein of a horse without any result being observed. Into the jugular vein of another horse, 30 grains of sulphate of solanin were injected. The animal was suddenly attacked with severe difficulty of breathing, and exhibited a great increase in the action of the heart, with convulsions, so that he appeared to be dying. In the course of twenty minutes he appeared quite recovered.

Two grains of acetate of solanin were injected into the rectum of a rabbit. The symptoms produced were heaviness, apathy, and slowness of movement: dilatation of the pupil followed, increased activity of the circulation and respiration, and convulsions, which, however, ceased in about two hours' time. The animal now moved but little, but when it did attempt locomotion, it dragged itself along with difficulty; but no special loss of power of the hinder extremities was observed. It died in six hours.

To a second rabbit, a certain quantity of acetate of solanin was given daily by the mouth, commencing with one grain. After several days, no effect being produced, the dose was increased to 2, and after another interval, to 3 grains. Some days after this the appetite lessened, but no other alteration being observed, the dose was increased to 4 grains. At last, the animal became heavy, slow in its movements, remained lying the greater part of the day, and at last died, without paralysis or any considerable fever having been observed.

Dr. Fraas is inclined to the opinion, that the pernicious effects which often follow feeding upon raw potatoes and potato-stalks, are due to the great quantity of alkaline and earthy salts which they contain, and never to the solanin. An analysis of the potatoes used in his experiments, gave the following results: In 100 parts of dried potatoes there were 4.22 per cent. of ash; or in 100 parts of fresh potatoes, 1.17 parts of ash. 100 parts of ash contained—

Sulphuric acid	2.90
Phosphoric acid	12.37
Silicic acid	A trace.
Chlorine	4.23
Potash	52.23
Soda	A trace.
Alumina	A trace.
Oxide of soda	A trace.
Magnesia	2.41
Lime	3.68
Carbonic acid	20.18
Loss	2.0
	100.00

The potatoes contained no solanin.—*Brit. and For. Med.-Chirurg. Rev.* July, 1854, from *Archiv. für Pathol. Anat. und Physiol. und für Klinische Medicin*, Bd. vi. Heft 2.

44. *Causes of Death from the Use of Anæsthetic Agents.*—Dr. K. King relates (*Edinburgh Med. and Surg. Journ.* Jan. 1854) a case of death occurring forty hours after the administration of chloroform, and makes the following remarks on the causes of death from the use of anæsthetic agents, which are worthy of consideration:—

"The subject of anæsthetic agents is one comparatively new to the profession; and although its rapid adoption and speedy admission as one of the recognized steps of every very important surgical operation, seem to disprove that slowness to leave the beaten track which has often been objected to against medical practitioners, it must be admitted that there still is a division of opinion as to the extent of their applicability, and the amount of danger which attends their use. In the opinion of some, whenever pain is to be inflicted, these agents may be employed, unless special circumstances, as diseased heart,

&c. contraindicate their use. Others, again, would limit their application to the severest surgical operations, considering that a certain amount of danger always attends their administration; and that therefore they are inapplicable in all cases which are usually painful, but unattended with fatal results. It is admitted by all, that deaths have not unfrequently occurred after the use of anæsthetic agents; but, according to some, the deaths actually caused by these agents are very rare, some of them being caused by accidental circumstances, and others by a neglect of those means which, if applied in time, would have obviated the fatal result. On inquiring into the mode in which death is produced, we find a remarkable discrepancy of opinion. In a very excellent paper in the September number of the *Edinburgh Monthly Journal* by Mr. Bickersteth, several cases and experiments are adduced to prove that death takes place usually, if not universally, by asphyxia. He gives as the result of his observations, that 'the respiratory movements cease before the cardiac;' and that, 'if artificial respiration be resorted to before the cardiac contractions are seriously affected, and be properly maintained for a sufficient period, the respiratory functions may be re-established.' He states it as his opinion that 'the pulse should not be taken as any guide during the administration of chloroform. It should be wholly disregarded, except under certain circumstances.' The attention should be mainly directed to the respiration; and, without denying the possibility of exceptions owing to peculiar idiosyncrasies, he considers that while the respiratory act is regularly performed, the state of anæsthesia may be produced without incurring any danger; but if the exhibition of the agent be persevered in while that state continues, syncope and asphyxia may occur simultaneously and cause sudden and irreparable death. But the fact of the pulse becoming less frequent is of no consequence—indeed is 'so constant, that by some it is supposed, and correctly supposed, that anæsthesia is not sufficiently complete for the performance of surgical operations, unless the pulse be below its standard.' Such at least is the general impression which his very admirable paper conveys to my mind; and if we can subscribe to his opinion, the induction of anæsthesia loses almost all its terrors, for death is preceded by a period of warning when, by prompt and simple measures, the fatal termination may be averted. The patient is in the condition of one seemingly drowned, but still capable of resuscitation. But unfortunately we find a different theory and different practice as decidedly and as ably recommended. In the *Lancet* of November 19, 1853, there is an abstract of a paper by M. Jobert De Lamballe, in which he says that 'the administration of chloroform should cease immediately when the beats of the heart decrease in number and force;' and again, 'the action of the heart should always be our guide; it is the best means of ascertaining the saturation of the nervous system by chloroform, and of judging how far this system has been influenced by the anæsthetic agent.' As to practice, M. Jobert 'prefers acting principally on the nervous system, rather than lose time by exciting the functions of respiration and circulation.'

"Without going at any length into this matter, I believe that there is much greater probability of asphyxia than of syncope supervening from the use of chloroform; because the muscles of respiration (and consequently the respiratory act) are governed by nerves connected with the cerebro-spinal system, which is much more readily affected by this agent than are the sympathetic nerve and its branches, which govern the cardiac movements, and which we have no reason to suppose are affected except secondarily. It is therefore natural to conclude that in most cases the failure of the respiratory act will take place before that of the heart; and that on its being noticed and the proper means adopted, time will be given to arrest the circulation of venous blood in the arteries, the continuance of which would necessarily prove fatal. At the same time, I fear we must consider it as proved that syncope may supervene without this premonitory notice. In a case recorded by Dr. Dunsmure in the November number of the *Monthly Journal*, the breathing was not observed to fail before the pulse stopped. Those 'who had an opportunity of observing the respiration, positively assert that the breathing did not cease before the pulse.' It is stated that after 'artificial respiration was had recourse to, in a

few moments he made a long inspiration, which was followed by four others at gradually lengthening intervals,' while 'no pulsation could be felt in the radial arteries.'

"An anonymous correspondent of the *Medical Times* (October 15) concludes from these facts that this was a case of 'mere syncope,' and that therefore, as a rule, 'the state of the pulse should be attended to. as betraying the first symptom of impending danger.' But it must be remembered that in such cases every inspiratory act is necessarily observed, while we have no clear evidence of the last contraction of the heart: the mere fact of the pulse at the wrist being imperceptible is no evidence that the heart had not contracted; and I have some difficulty in believing that five acts of inspiration took place without a single contraction of the heart! I believe, in this instance, that a failure of the heart's action (syncope) and a paralysis of the respiratory muscles (leading to asphyxia), occurred at the same instant—that a partial reaction took place, when most probably the observed acts of inspiration were accompanied by unobserved contractions of the heart—but that the efforts of nature were incapable of setting the machinery in motion, and that from the last natural inspiration the man's death is to be dated. The anonymous writer before referred to seems to think that this event is to be dated from the last perceived pulsation; but it would be monstrous to apply the term death to a man who was still capable of making natural respiratory efforts; and, if it were as capable of demonstration, I believe that in this case contractions of the heart would have been found taking place after the last act of inspiration. But I do not deny that deaths have occurred in which failure of the heart's action has been the prominent symptom, and that syncope has been attended by its usual symptoms of pallor of the countenance, &c. (In Dr. Dunsmure's case the face and heart are said to have been a good deal congested.) Two cases are mentioned in the Dublin *Medical Press* (April 20, 1853), in which syncope having supervened, M. Nelaton restored suspended animation by inverting the body, after which the face recovered its colour, and the cases terminated favourably. And in a case of M. de Vallet, in the Hôtel Dieu d'Orleans, the patient became suddenly pale, respiration ceased, and tracheotomy, artificial respiration, and galvanism were of no avail to prevent the fatal result. I conceive, then, that it is clearly established—1. That a continuance of the use of anæsthetics would in all cases, if sufficiently long employed, paralyze all the muscles supplied by the cerebro-spinal axis, including the muscles of respiration; and, secondarily, those dependent on the sympathetic system; that in all cases an overdose of chloroform may be expected to produce asphyxia, which, however, may, in most cases at least, be arrested, if observed before the cessation of the heart's action, by the methods usually employed. 2. That in some cases, altogether independently of organic disease, the heart's action may suddenly cease, even without an undue quantity of the drug having been employed; when, if the heart can be stimulated to contract, respiration (which cannot in the living body go on independently of the heart's action) may be restored and life preserved. The treatment here would be that usually adopted in syncope; for if the heart can be stimulated to contract, the respiratory muscles are capable of performing their functions; the patient is simply in the condition of one who has fainted. This class of cases must be rare, because anæsthetics (as before remarked) act on the cerebro-spinal much more readily than on the sympathetic system. 3. That in another class of cases death is produced by the simultaneous failure of the respiratory and cardiac movements; that these are the most dangerous of all; and that the treatment employed ought to combine the methods used in the first two cases.

"At one time I thought that death always resulted from asphyxia; but the reported cases before alluded to, and the observations both of myself and others, convince me that we must be prepared to meet with other and even more formidable dangers. However much this is to be regretted, I think it must now be admitted, and ought to be generally known; for if the profession and the public are to have the full benefit of anæsthetic agents, it is necessary to look the dangers and difficulties attending them fairly in the face, when, by a proper discrimination of the causes likely to lead to a fatal result, we may be

prepared *promptly* to apply those remedies more particularly suited to the case in question. We must be ready to lay down preconceived views and prejudices; and no man need be ashamed to change his opinion on a subject still new to the profession, and regarding which every day is accumulating experience and elucidating new facts.

"But to return to my case, from which I have wandered further than I intended: it is quite clear that by none of these methods was death produced. The administration of chloroform presented no peculiarities; he recovered from its effects in the usual way. When I saw him ten hours after, there was nothing about him to induce the slightest apprehension. Are we therefore to exonerate the chloroform from any share in the fatal result? Is the fact of a perfect recovery from the immediate effects of the anæsthetic agent to be received as proof that the system had entirely thrown it off, and that the condition of the individual is precisely what it was before its administration? The symptoms which on the 2d September manifested themselves, bore too strong a resemblance to those artificially induced on the 1st, not to suggest the idea that the two events were in some way connected. There was the loss of the intellectual powers—diminished sensibility, the strongest sinapisms produced no appearance of uneasiness—the respiration was impeded and stertorous—in a word, the condition in which he was on the 2d, seemed only an exaggeration of that produced on the 1st. Indeed chloroform acts by suspending the powers of the cerebro-spinal axis; its influence being directed especially to the sensitive nerves. Could we find an agent whose actions are limited to this, we would have reached the acme of anæsthetic discovery. In addition to the fatal results of asphyxia and syncope, are we to add to the dangers of administering chloroform those which may arise from a return of coma? Is it possible that the comatose state induced by chloroform may outlast the period of its administration, or even return after some hours of apparent health? This question is one which has often forced itself on my mind, and to which the mere fact of no such occurrence having ever taken place seemed hardly a sufficient answer. Convulsions or epileptic fits seem not uncommon results. Dr. Dunsmure, in speaking of his fatal case, remarks, that 'he seemed to take a slight convulsion like an *epileptic fit*, and *such as I have seen on several occasions* in people who have led an intemperate life.' Dr. Murphy, in the *Association Journal*, September 2, 1853, details two cases in which chloroform was administered during parturition; in the first of which 'distinct dyspnœa, excessive lividity of the face, and all the signs of extensive engorgement of the lungs and head' came on an hour and a half after labour; and though temporarily relieved, half an hour after 'she suddenly awoke with a return of the distressing dyspnœa, that was soon followed by *convulsions* and almost immediate death.' In the second case, two hours after the termination of labour, the patient was seized with slight cough and some difficulty of breathing: in the evening, dyspnœa had so much increased, that she was cupped to 10 ounces. She slept for two hours, 'but when she awoke the dyspnœa returned with still greater distress, and continued to increase during the night. On the following morning she became asphyxiated, and died about 2 o'clock P. M.,' 24 hours after the termination of labour. In remarking on these cases, Dr. Murphy asks the very appropriate question: Is it probable that, when no dangerous symptoms presented themselves during the administration of chloroform, fatal effects could result from it 'at a time when the vapour was dissipated, or, if any remained, its force greatly weakened, and less likely to cause such effects?' We know that the chloroform acts on the nervous centres, and that its action is usually evanescent; but is it necessarily so? After the cause has been removed, may not the effect remain? The second of Dr. Murphy's cases I have sometimes thought to have a shade of resemblance to the one I have just detailed, as far as symptoms go. The account is too scanty to enable us to form a decided opinion on this subject. Dr. Murphy states that she died asphyxiated. Now my patient died asphyxiated, *i. e.* the heart beat rapidly and convulsively after the respiration had become almost impeded; but I do not consider asphyxia to have been the *cause* of death, because, though its immediate precursor, it came on merely in the train of events. Death commenced from the brain (coma), and extended

to the nerves of respiration (asphyxia). Had there not been *post-mortem* appearances clearly pointing to different causes of death, one might have thought that in both cases the fatal supervention of paralysis of the nervous centres might have been prepared by its first artificial induction. Dr. Murphy states that it is improbable this should occur, seeing that he cannot 'find the report of a single case in which the patient who inhaled chloroform without an unfavourable symptom was attacked two hours after with dyspnœa, still later became asphyxiated and died.' And again he states that 'he cannot find a case of the kind in the whole range of surgical practice.'—'The danger—the only danger—which the surgeon dreads, is sudden death during the operation.' I do not mean to say, that in Dr. Murphy's cases death was caused by chloroform; but I do think that we have a right, derived from *à priori* reasoning, to fear that all danger is not past when the patient emerges unharmed from the anæsthetic condition; and that it is only more extended observation which can prove whether those fears are wholly chimerical. My own impression is, that in Mr. T.'s case death was not caused by the administration of chloroform; but I ground that opinion, not on the fact that no other case of the sort has been recorded, nor on the supposition that, in the nature of things, such an event might not occur, but on the appearances presented by the *post-mortem* examination, coupled with the symptoms for which he first sought my advice.

"A comatose condition was induced by chloroform, which was as evanescent as that state so induced commonly is—twenty-four hours afterward true coma supervened, and proved fatal. But the *post-mortem* examination of the head showed that this latter condition was connected with effusion of bloody serum—a common cause of coma, but not (as far as we know) ever observed as a result of chloroform. In cases where death has resulted from the employment of this agent, effusion has never been found, and in many it is distinctly mentioned that the brain was not even congested; so that the comatose state is produced without the intervention of vascular excitement, and therefore by a process different from that which was found to have occurred in the case I have related. I admit, however, that it is possible that an influence may be exerted on the brain, capable of superinducing, after a longer or shorter period, effusion of serum or other secondary effects; as we know that a person may recover from concussion, and yet sink under other results of the same cause which produced the concussion. Nor will this appear a far-fetched comparison, if we suppose, as I think we must do, that chloroform acts directly on the nervous centres. But there is a feature of the *post-mortem* examination which I now would call attention to, and that is the pus found between the parietal and visceral layers of the arachnoid. This is a rare phenomenon, and therefore not so well understood as other lesions of the brain and its membranes, which have been more frequently observed. It was found as a thin layer spread over the summits of both lobes, and adherent to the visceral portion. It did not extend to the basilar portions of the brain, and therefore can hardly be supposed to have been caused by any extension of inflammation from the mucous membrane of the nose. Indeed, neither during life nor after death was there anything to indicate that inflammation of the Schneiderian membrane had taken place. It could not have caused compression, and therefore is of importance only as proving the existence of previous arachnoid inflammation. But there was no redness or visible sign of inflammation of the arachnoid. There was much congestion of the cerebral vessels, but the products of their inflammation would have been thrown out in the subarachnoid tissues. Now I cannot help thinking that had there been so intense inflammation of the arachnoid as to have caused purulent effusion in the space of time between my visit at ten o'clock P. M. and his death twenty-seven hours after, there would necessarily have been some *post-mortem* evidence. I conceive, therefore, that the condition which led to the formation of pus was pre-existent to the administration of chloroform. The feeling of stupidity, difficulty of articulation, loss of sense of smell, &c., may all have resulted, not from the closure of the nares by polypi, but from this internal disease. There was no heat of head—no apparent increased vascular action of any sort—nothing to call attention to any other cause than the mechanical obstruction. But after calmly weighing the whole

facts of the case, I firmly believe that a low obscure form of arachnoid inflammation produced those symptoms which seemed fairly and naturally to be referred to the presence of polypi. What effect the administration of chloroform may have had in such circumstances it is difficult to say, but I feel convinced that it at all events did not set up the action which terminated so fatally.

"The *post-mortem* appearances in this case were very peculiar. The position of the pus on the free surface of the arachnoid must be held conclusive as to inflammation having existed in that membrane. Dr. Watson mentions the fact of such effusion as being rare (vol. i. p. 380), and quotes from Dr. Abercrombie a case in which it was observed in a child who had been three weeks ill; and says, that 'if simple arachnitis of an acute kind ever happen, it has not been my fortune to see or to recognize it.' While pus was found in the summit of the brain, the effusion of serum at the base was into the arachnoid cavity, and not in the subarachnoid space; and though bloody (*i. e.* red-coloured) serum was found in the ventricles in more than usual quantity, it was not excessive, nor by itself at all sufficient to cause death. There was also congestion of the bloodvessels of the brain; not inflammation of its substance. But that and the effusion of serum might, and probably did, take place within the last twenty-four hours of life—the purulent deposit I think hardly could.

"On these grounds I maintain that in the case I have related death did not result from chloroform. At the same time, I give it as my decided opinion, that until we find an agent which is capable of producing anæsthesia—inaction of the nerves of sensation—without implicating the functions of the cerebrum and motor nerves, we must be prepared to encounter occasional dangerous cases of asphyxia and syncope, and fatal ones in which these states occur simultaneously; and further, I would be inclined to expect, that cases might yet occur to prove that with the cessation of the immediate effect of the anæsthetic agent danger has not entirely ceased. In the meantime, I think it the duty of every surgeon to communicate to the profession any fact occurring in his practice in relation to this subject which presents novel and interesting features. It is in this way only that we shall learn the true merits of the case, and decide the important question of the compatibility of the anæsthetic agents at present known with all constitutions free from actual organic disease."

45. *Detection of Blood-stains on a Knife covered with Rust.*—M. Daubrawa was requested to ascertain the existence of blood-stains on a knife which was suspected to have been used in the commission of a murder. The knife having lain a long time in a damp place, was rusted; but certain bright rust-free spots could be distinguished amid the rust. On heating the point of the blade, these spots scaled off, while the rust remained adherent; on the other hand, on immersing the knife in dilute hydrochloric acid, the bright spots remained untouched, although the rust was readily dissolved. It was probable that these bright spots were blood-stains; but as some non-nitrogenous organic acids will produce similar marks, some of the detached scales were heated in a test-tube, and, by the disengagement of ammonia from the hematine of the blood, caused a blue colour on reddened litmus-paper. The whole blade was then macerated for a long time in distilled water, which acquired a reddish discoloration, and by the aid of a lens, fibrin could be seen adhering to the blade in the situation of the bright spots. Ammonia added to the solution caused no precipitate; nitric acid gave a white precipitate; it became turbid from heat; solution of chlorine at first produced a green tint, this colour then disappeared, and white flocculi were deposited. These different fluids having been evaporated to dryness and burnt, and the residue dissolved in hydrochloric acid, demonstrated the presence of iron by its appropriate reagents.—*Journal de Chimie Médicale*, December.